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Term:

L4 and gene near therap\$ and (diabetes or
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<u>L6</u>	L4 and gene near therap\$ and (diabetes or hypertension or parkinson or retinopathy or huntington)	19	<u>L6</u>
<u>L5</u>	L4 and gene near therap\$	39	<u>L5</u>
<u>L4</u>	(superoxide near dismutase\$ or SOD or "SOD-1" or CuZn near SOD or CuZnSOD) near10 treat\$ and adenovir\$	57	<u>L4</u>
<u>L3</u>	(superoxide near dismutase\$ or SOD or "SOD-1" or CuZn near SOD or CuZnSOD) and adenovir\$	1045	<u>L3</u>
<u>L2</u>	(superoxide near dismutase\$ or SOD or "SOD-1" or CuZn near SOD or CuZnSOD) near20 adenovir\$	23	<u>L2</u>
<u>L1</u>	(superoxide near dismutase\$ or SOD) near10 adenovir\$	18	<u>L1</u>

END OF SEARCH HISTORY

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Terms	Documents
L4 and gene near therap\$	39

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set hi ;set hi
HILIGHT set on as ''
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? begin 5,6,55,154,155,156,312,399,biotech,biosci
>>>      135 is unauthorized
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Set	Items	Description
? s (superoxide (n) dismutase or SOD or "SOD-1" or CuZnSOD or CuZn (n) SOD) (5n) adenovir?		
Processed 30 of 34 files ...		
Completed processing all files		
	331148	SUPEROXIDE
	206302	DISMUTASE
	204410	SUPEROXIDE(N) DISMUTASE
	84895	SOD
	166	SOD-1
	2838	CUZNSOD
	6156	CUZN
	84895	SOD
	2439	CUZN(N) SOD
	193750	ADENOVIR?
S1	439	(SUPEROXIDE (N) DISMUTASE OR SOD OR "SOD-1" OR CUZNSOD OR CUZN (N) SOD) (5N) ADENOVIR?

? s s1 not py>1995
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Processing
>>>One or more prefixes are unsupported
>>> or undefined in one or more files.
Processed 20 of 34 files ...
Processing
Completed processing all files

439 S1
46642802 PY>1995
S2 13 S1 NOT PY>1995

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DIALOG(R)File 5:Biosis Previews(R)
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04748390 BIOSIS NO.: 000080051517
MODIFICATION OF DOPA TOXICITY IN HUMAN TUMOR CELLS
AUTHOR: PARSONS P G
AUTHOR ADDRESS: QUEENSLAND INSTITUTE OF MEDICAL RESEARCH, HERSTON,
QUEENSLAND, AUSTRALIA 4006.
JOURNAL: BIOCHEM PHARMACOL 34 (10). 1985. 1801-1808. 1985
FULL JOURNAL NAME: Biochemical Pharmacology
CODEN: BCPCA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: A variety of factors were found to modify the toxicity of L-dopa in HeLa cells (D37 16 .mu.M) and in dopa-sensitive, nonpigmented human melanoma cells (MM96) (D37 5 .mu.M) having a similar size and doubling time. Dopa toxicity was decreased by concurrent treatment with superoxide dismutase, peroxidase or catalase, by erythrocytes, or by hypoxia.

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Toxicity could be increased by the enzyme inhibitors L- and D-penicillamine, sodium diethyldithiocarbamate or 3-amino-1,2,4-triazole. The 2 cell lines had similar levels of superoxide dismutase and peroxidase; in 6 human melanoma lines, no correlation was found between dopa killing and tyrosinase activity as determined either by formation of

dopa from tyrosine or by formation of melanin from dopa. Uptake of L-dopa was similar in HeLa and MM96 cells, and the toxicity of D-dopa was the same in both lines as that of the L-isomer. Dopa decomposed within 12 h in culture medium, the rate and products being influenced by addition of the above enzymes and by the cell density. Dopa-melanin and medium containing decomposed dopa were also selectively toxic to MM96 cells. Adenovirus 5 was used in 2 different ways to assess the relative importance of DNA damage and inhibition of DNA synthesis by dopa. Viral replication was found to be unaffected in cells being treated with dopa but was strongly inhibited in cells treated with the DNA polymerase inhibitor cytosine arabinoside. Secondly, the virus was itself inactivated by treatment with dopa for 24 h (D37 1.3 mM); similar dose

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response curves were obtained for replication of dopa-treated virus in untreated HeLa or MM96 cells. These results show that the initial events of dopa toxicity occur outside the cell and lead to the formation of a stable, toxic product (probably melanin) which does not strongly inhibit DNA polymerase activity. Melanoma hypersensitivity was not due to differences in oxygen-metabolizing enzymes, dopa uptake, or DNA repair.

DESCRIPTORS: MELANOMA CELLS HELA CELLS L PENICILLAMINE D PENICILLAMINE
SODIUM-DIETHYLDITHIOCARBAMATE 3 AMINO-1 2 4-TRIAZOLE **SUPEROXIDE**
DISMUTASE PEROXIDASE **ADENOVIRUS** 5 DNA DAMAGE

CONCEPT CODES:

02508 Cytology and Cytochemistry-Human
10808 Enzymes-Physiological Studies
13012 Metabolism-Proteins, Peptides and Amino Acids
22005 Pharmacology-Clinical Pharmacology (1972-)
22504 Toxicology-Pharmacological Toxicology (1972-)
24008 Neoplasms and Neoplastic Agents-Therapeutic Agents; Therapy

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10010 Comparative Biochemistry, General
10012 Biochemistry-Gases (1970-)
10060 Biochemical Studies-General
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
11108 Anatomy and Histology, General and Comparative-Microscopic and
Ultramicroscopic Anatomy
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
22003 Pharmacology-Drug Metabolism; Metabolic Stimulators
33506 Virology-Animal Host Viruses

BIOSYSTEMATIC CODES:

02210 Adenoviridae (1979-)
86215 Hominidae

BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA):

Microorganisms
Viruses

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Animals
Chordates
Vertebrates
Mammals
Primates
Humans

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DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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00976947 Genuine Article#: FK380 Number of References: 36
Title: THE E1B ONCOGENE OF ADENOVIRUS CONFERS CELLULAR-RESISTANCE TO
CYTOTOXICITY OF TUMOR-NECROSIS-FACTOR AND MONOCLONAL ANTI-FAS ANTIBODY
Author(s): HASHIMOTO S; ISHII A; YONEHARA S
Corporate Source: MEIJI INST HLTH SCI, 540 NARUDA/ODAWARA 250//JAPAN/; TOKYO
METROPOLITAN INST MED SCI, DEPT CELL BIOL, BUNKYO KU/TOKYO 113//JAPAN/
Journal: INTERNATIONAL IMMUNOLOGY, 1991, V3, N4, P343-351
Language: ENGLISH Document Type: ARTICLE
Geographic Location: JAPAN
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: IMMUNOLOGY
Abstract: The cell lines KB8, 16, and 18 are KB cells which constitutively
express adenovirus type 2 (Ad2) Ela, Ela plus Elb, and Elb genes,
respectively. We show here that KB18 cells are completely resistant to
cytolysis by tumor necrosis factor (TNF) or anti-Fas, although KB8 and
KB or KB16 are highly and moderately sensitive, respectively. The

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levels of receptors for TNF and anti-Fas of KB18 were almost the same
as compared with those of KB or other KB-cell lines. Expression of
manganous superoxide dismutase (MnSOD) mRNA in KB18 was about 20-fold
higher than that in KB or KB8 cells. KB, HT29, and A673 cells infected
with dl337 (an Ad5 mutant defective in Elb function) are highly
sensitive to TNF or anti-Fas, although wild-type Ad2-infected cells are
resistant. Our results indicate that the Elb oncogene can confer
cellular resistance to cytolysis by either TNF or anti-Fas in both KB
cells and adenovirus-infected human cell lines through influencing
intracellular events including regulation of MnSOD genes. Furthermore,
we describe how anti-Fas mimics only the cytolytic activity of TNF,
whereas TNF also has many other biological activities.
Descriptors--Author Keywords: **ADENOVIRUS** E1A GENE; MANGANOUS
SUPEROXIDE DISMUTASE; FAS-ANTIGEN; KB CELLS
Identifiers--KeyWords Plus: MANGANOUS SUPEROXIDE-DISMUTASE; CYTO-TOXICITY;
FACTOR TNF; BINDING; RECEPTORS; CELLS; HOST; GENE; DEGRADATION;
EXPRESSION

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DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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Research Fronts: 89-0312 003 (TUMOR NECROSIS FACTOR; ANTIVIRAL ACTIVITY;
EFFECTS OF CYTOKINES)
89-7486 001 (TUMOR NECROSIS FACTOR; LANGERHANS CELLS IN VACCINIA
VIRUS-INFECTION; INTERFERON-GAMMA BINDING)

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TSUJIMOTO M, 1985, V82, P7626, P NATL ACAD SCI USA
VANHAESEBROECK B, 1990, V176, P362, VIROLOGY

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WHITE E, 1984, V52, P410, J VIROL
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WONG GHW, 1986, V323, P819, NATURE
WONG GHW, 1988, V242, P941, SCIENCE
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YOSHIDA K, 1987, V1, P645, GENE DEV

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Display 2/9/3 (Item 1 from file: 266)

DIALOG(R)File 266:FEDRIP

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00351692

IDENTIFYING NO.: 3P01NS37520-03S1 9002 AGENCY CODE: CRISP

Core--Vector

PRINCIPAL INVESTIGATOR: SAPOLSKY, ROBERT M

ADDRESS: STANFORD UNIVERSITY MED CTR R 161 STANFORD, CA 94305-5327

PERFORMING ORG.: STANFORD UNIVERSITY, STANFORD, CALIFORNIA

SPONSORING ORG.: NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE

FY : 2001 TYPE OF AWARD: Supplement (Type 3)

SUMMARY: The Vector core will produce herpes simplex virus (HSV)-based amplicon vectors, adenoviral vectors, and retroviral vectors. These will be used in Project 1 in mixed neuronal/glial cultures and pure astrocyte cultures, as well as in Project 2, for in vivo ischemia studies. HSV and adenoviral vectors will be generated expressing the genes for CuZn-superoxide dismutase (SOD1), glutathione peroxidase (the prior two either individually, or in combination, Bcl-2, and Hsp70. All of the HSV and adenoviral vectors will be "bicistronic", expressing both the gene in

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DIALOG(R)File 266:FEDRIP

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question, as well as a reporter gene. For each experimental vector, the cognate control vector will contain the same gene in question with a stop codon inserted in its center, insuring non-expression. Similar controls will be generated for the adenoviral vectors. Retroviral vectors to express SOD1, Mn-SOD (SOD2), Bcl-2 and Hsp70 will be produced. Control vector will express either the reporter gene beta-galactosidase or a stop codon version of the gene of interest. The retroviral vectors for HSP70 and Bcl-2 have been constructed and they will be produced in the vector core. In addition new vectors to express SOD1 and SOD2 (separately) will be constructed and then produced. The purpose of the Vector Core will be to ensure a constant supply of vectors for the various groups, to insure quality control in the production of such vectors, and to troubleshoot problems, as they arise, in individual laboratories in the use of vectors.

DESCRIPTORS: biomedical facility; **superoxide dismutase**; stress protein; **Adenoviridae**; Alphaherpesvirinae; Retroviridae; transfection /expression vector; BCL2 gene /protein

- end of record -

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00351689

IDENTIFYING NO.: 3P01NS37520-03S1 0002 AGENCY CODE: CRISP

In vivo injury paradigms

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SPONSORING ORG.: NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE

FY : 2001 TYPE OF AWARD: Supplement (Type 3)

SUMMARY: Recent progress in the area of stroke research suggests that a number of molecular mechanisms are intimately involved in the evolution of ischemic brain injury. Gene induction has been observed following ischemia, but the exact roles of many are not yet well known. Some gene products are known to be detrimental to the cell while others are felt to be neuroprotective. The goals of this project are to define more precisely the roles of three classes of genes which may play neuroprotective roles. They are: the proto oncogene, bcl-2, antioxidant genes (sod-1 and gspx) and the

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stress protein, hsp70. In Project 2, we will utilize genetically normal animals and study 3 different in vivo models of ischemia (2 focal and one global models of cerebral ischemia). We will alter gene expression via gene

transfer using defective herpes simplex and adenoviral vectors. We will study the limits and conditions under which gene over-expression may improve neuron survival. We hypothesize that injury due to some, but not other kinds of insults will be attenuated with gene product over-expression, and that these observations will offer insight into the pathophysiology of cell death. We will examine whether gene transfer after the onset of injury is neuroprotective, and whether over-expression of Bcl-2 and antioxidant genes are protective against permanent as well as transient focal cerebral ischemia. We will also examine mechanisms underlying neuroprotection, or lack of neuroprotection, by examining the participation of other gene products such as the stress proteins, caspases and Bcl-2 family proteins in response to gene over-expression and cerebral injury. We will also study whether gene over-expression alters generation of superoxide and apoptosis. These novel approaches will hopefully add

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insight into the complex molecular processes involved in cerebral ischemia and may lead to the development of treatments for stroke and other degenerative disorders.

DESCRIPTORS: laboratory rat; cell death; cerebral ischemia /hypoxia; transfection; protooncogene; gene induction /repression; immunocytochemistry; disease /disorder model; electron microscopy; antioxidant; **superoxide dismutase**; stress protein; **Adenoviridae**; Alphaherpesvirinae; transfection /expression vector; transient ischemic attack; neuroprotectant; neuropathology; BCL2 gene /protein

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00351688

IDENTIFYING NO.: 3P01NS37520-03S1 0001 AGENCY CODE: CRISP

In vitro injury paradigms

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FY : 2001 TYPE OF AWARD: Supplement (Type 3)

SUMMARY: The protective potential of three groups of genes will be studied in ischemia-like injury of brain injury of brain cells from cortex, hippocampus, and striatum, in primary culture. First an antioxidant strategy will be tested by over-expressing CuZn **superoxide dismutase** (SOD1) using herpes virus and **adenoviral** vectors to achieve rapid expression in neurons and astrocytes, and retroviral vectors for prolonged, stable expression in astrocytes. Whether acute expression can provide protection will be tested. If this is not protective, the

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effect of prolonged stable expression and the use of bicistronic vectors, to rapidly express both SOD and a downstream antioxidant vectors, to rapidly express both SOD and a downstream antioxidant enzyme, will be tested. We will test for protective effects, as well as for induction of

other antioxidant enzymes. Whether protection correlates with induction of other antioxidant enzymes will be determined. Under conditions where protections seen the extent of oxygen radical production, lipid peroxidation and changes in level of glutathione will be determined. Whether this gene protects against necrotic or apoptotic forms of cell death will be determined. Whether this gene protects against necrotic or apoptotic forms of cell death will be determined, as well as the time window in which expression can still protect, since it is important to develop therapeutic strategies that are effective after insults. Second, we will study the ability of Bcl-2 expression to protect in the same injury paradigms. Oxidative status and the time window in which Bcl-2 can protect will be determined. Third we will study the ability of the inducible heat shock protein 70 (HSP70) to protect from these injury again analyzing the

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time window during which this gene can protect and whether it blocks apoptotic or necrotic cell death. Primary cultures are particularly useful for analyzing mechanisms of ischemic brain injury and mechanisms of protection at the cellular level. Primary cultures of neurons and glial cells and pure astrocyte cultures will be made from hippocampus and striatum. Results will be compared with parallel studies carried out on primary cultures from neocortex. In addition, astrocyte cultures will be produced from transgenic mice made with different gene dosages of SOD1 or MnSOD (SOD2), including knockouts lacking these genes, to determine the importance of these enzymes for astrocyte survival of ischemia-like insults. These studies will provide fresh insight into possible mechanisms of protection by three candidate genes for anti-ischemic gene therapy tested in three brain regions.

DESCRIPTORS: laboratory mouse; transgenic animal; hippocampus; occipital lobe /cortex; brain cell; cell death; cerebral ischemia /hypoxia; enzyme induction /repression; gene therapy; disease /disorder model; glia; astrocyte; antioxidant; **superoxide dismutase**; stress protein;

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tissue /cell culture; **Adenoviridae**; Alphaherpesvirinae; transfection /expression vector; neuroprotectant; BCL2 gene /protein

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00341002

IDENTIFYING NO.: 5R01HL60132-03 AGENCY CODE: CRISP

LUNG RADIATION PROTECTION BY MNSOD--TRANSGENE THERAPY

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SPONSORING ORG.: NATIONAL HEART, LUNG, AND BLOOD INSTITUTE

FY : 2001 TYPE OF AWARD: Noncompeting Continuation (Type 5)

SUMMARY: The lung is a major dose-limiting tissue in radiation therapy. We have demonstrated in mice that intratracheal infection of plasmid

liposomes or second generation replication-defective **adenovirus** constructs carrying the human manganese **superoxide dismutase** (MnSOD) transgene results in a significant decrease in both acute and chronic (alveolitis/fibrosis) damage by whole lung irradiation. Both

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delivery systems demonstrated increased MnSOD mRNA levels in the airway prior to irradiation and decreased levels of transcripts for inflammatory cytokines that irradiation. We will now confirm the observations, and optimize in rigorous preclinical studies the efficiency and safety of pulmonary MnSOD transgene therapy using plasmid/liposomes for lung irradiation protection. Three specific are designed for proof of the principal that MnSOD transgene therapy will protect normal lung from irradiation damage. In the first specific aim C57BL/6J mice, which are heterozygous deletion recombinant-negative for murine MNSOD, will be compared will be compared with normal littermates for irradiation-induced organizing alveolitis. Our preliminary data show increased susceptibility of MnSOD heterozygous knockout mice to irradiation-induced alveolitis and are corroborated by the increased radiosensitivity in vitro of (-/-) and (-/+) human MnSOD transgene. In the second specific aim we will determine the level of over- expression of MnSOD which correlates with the relative lung radioresistance in FeVB/NHsd transgenic mice over-expressing the human MnSOD transgene in the airway linked to the lung parenchymal cell-specific